Mekanisme dan algoritme diagnostik aritmia atrium kanan

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Abstrak

Latarbelakang: Kondisi atrium kanan yang terdiri dari berbagai struktur yang kompleks menyebabkan timbulnya variasi sifat elektroiisiologis yang memberikan kemudahan timbulnya aritmia. Aritmia atrium kanan merupakan jenis aritmia yang panting karena prevalensi yang tinggi dan konsekunsi klinis yang berbahaya. Akan tetapi epidemiologi aritmia atrium kanan beserta karakteristik eleklrofisiologinya di Indonesia belum pemah dilaporkan. Krista terminalis yang merupakan garis hambatan konduksi posterior pada kepak atrium (KA), dan sumber trbanyak takikardia atrium (TA), nierupakan struktur unik dengan karakteristik elektrofisiologis yang belum diungkap secara luas. Di lain pihak, berkembangnya pemahaman mekanisme KA, menimbulkan masalah diagnosis karena adanya kemiripan morfologi gelombang kepak antar berbagai jenis KA yang mekanismenya berlainan, dan adanya variasi morfologi gelombang kepak pada KA yang sejenis. Oleh karena itu akan dilakukan rangkaian penelitian untuk menjawab beberapa masalah mekanisme dan diagnosis aritmia atrium kanan.

Metode: Dilakukan studi elektrofisiologi baik secara konvensional maupun dengan panduan sistem pemetaan non-kontak Ensite pada subyek dengan KA dan TA. Pada KA yang melibatkan ismus kavotrikuspid (KA-IKT) dilakukan entrainment untuk konfirmasi diagnosis. Pada ULR, lokasi dan lebar taut konduksi ditentukan atas dasar perubahan konvergensi propagasi impuls setelah melalui krista temiinalis. Pola aktivasi sumber TA dianalisis meinalcai propagasi impuls dan elektrogram unipolar virtual. Nilai 30% dari voltase negatif puncak dipakai sebagai pembeda daerah parut dari jadngan sehat. Analisis rnorfologi gelombang kepak pada EKG 12-sadapan dilakukan oleh dua orang ahli elektrofisiologi yang bebas. Suatu algoritme diagnosis KA yang sederhana akan dibuat
atas dasar EKG permukaan. Ablasi frekuensi radio (AFR) dilakukan pada sumber atau sirkuit reentry aritmia atrium kanan dengan memakai teknik yang sudah baku.

Hasil: KA tipikal merupakan kasus KA terbanyak di Pusat Jantung Nasional Harapan Kita, dan Iebih dari 60% subyek KA mempunyai penyakit jantung struktural. Rcrata panjang siklus takikardia (PST) ialah 261,8 ± 42,84, 226,5 ± 41,23, dan 195,4 ± 9,19 mdet masing-masing untuk KA tipikal, tipikal terbalik dan atipikal (p = 0,016). Morfologi EKG pada KA tipikal terdiri dari 3 tipe gelombang kepak yaitu F-/f+ di sadapan inferior dan P+ atau F+/f- di V, (tipe 1); F- di sadapan inferior dan P+ di V1 (tipe 2); dan f-/F+ di sadapan inferior dan F+ di V1 (tipe 3). Pada KA tipikal terbalik didapatkan 2 tipe rnorfologi yaitu P+ di sadapan inferior dan F- di V1 (tipe 1); dan P+ di sadapan inferior dan isoeiektrik di V; (tipe 2). Akan tetapi tidak didapatkan perbedaan bermakna aktivasi atrium kanan pada variasi morfoiogi KA-IKT. Tidak didapatkan konduksi transversal Krista terminalis pada 90% KA-IKT, sebaliknya didapatkan

konduksi transversal pada seluruh ULR. Pada saat ULR, KKL lebih cepat dari pada KK-r (1,228 ± 0,43 vs. 0,73 ± 0,30 m/det, p < 0,001). Rasio KK;/KKT ialah 1,95 ± 0,77 yang berbanding terbalik dengan lebar taut krista terminalis (1,57 ± 6,8 mm) (p < 0,00l).

Algoritme diagnosis baru atas dasar morfologi dan amplimdo gelombang kepak di sadapan I mempunyai akurasi 90 hingga 97%, sensitivitas S2 hingga 100% dan spesifisitas 95% dalam membedakan KA tipikal terbalik dari ULR. TA fokal mayoritas berasal dari krista terminalis dan memperlihatkan adanya jalur konduksi istimewa. Dengan teknik konvensional, keberhasilan AFR pada IKT, taut krista terminalis pada ULR dan TA fokal berturut-turut mencapai 96 % , 90% dan 91,7%.

Kesimpulan: KA tipikal merupakan KA terbanyak pada populasi penelitian ini, dengan mayoritas menderita penyakit janlung struktural. Tidak terdapat perbedaan aktivasi atrium kanan pada variasi morfologi gelombang kepak pada KA-IKT. Mayoritas taut konduksi krista terminalis bersifat fungsional dan selalu didapatkan pada saat ULR. Suatu algoritme diagnosis baru, akurat untuk membedakan KA tipikal terbalik dari ULR. Impuls TA fokal menyebar ke seluruh atrium setelah melalui jalur konduksi istimewa. AFR efektif menyembuhkan KA-IKT, KA non-IKT dan TA.;Background: Complex structures with variable electrophysiological properties in right
atrium facilitate arrhythmias occurrence. The right atrial arrhythmia is one of clinically important anrhythmias as it has high prevalence and significant clinical consequences. However, clinical and electrophysiological characteristics of iight atrial arrhythrnias have not been elaborated in Indonesia. The crista terrninalis has been shown as a posterior obstacle line during atrial flutter (AFL), and as a major source of focal atrial tachycardia (AT). However, as a unique structure of right atrium, little has been known about Crista terrninalis electrophysiological properties as a substrate of right atrial arrhythmias. A better understanding of AFL mechanisms yielded a diagnostic problem, since the flutter wave of different AFL has similar rnorphologies and the variable morphologies of the same AFL. Therefore, we conduct several interconnected study to overcome those diagnostic and mechanisms issues in right atrial arrhythmias.

Methods: Atrial flutter and AT subjects underwent electrophysiology study using conventional and/or noncontact mapping Ensite system. Entrainment pacing was performed to confirm the diagnosis of cavotricuspid isthmus (CTI) dependent AFL. In ULR subjects, location and width of gap conduction was determined by the change of convergent wavefront as it is passed the crista terminalis. Careful wavefront and virtual unipolar electrogram analysis was performed during focal AT. A value of 30% of peak negative voltage was used to differentiate low voltage zone and normal tissue. Two independent electrophysiologist analyzed the morphology and polarity of flutter wave in standard 12-lead ECG. Radiofrequency ablation was peformed at the origin and/or
reentry circuit of right atrial arrhythmias using a standard technique.

Results: Typical APL is predominant AFL cases in National Cardiovascular Center

Harapan Kita. More than 60% of all AFL cases suffered from structural heart disease. Mean tachycardia cycle length of typical, reverse typical and atypical AFLS were 261.8 ± 42.84, 226.5 ± 41.23, and 195.4 ± 9.19 msec, respectively (p = 0.0l6). Typical AFL

showed 3 types flutter wave morphologies comprised of F-/f+ at inferior and P+ or F+/f- at V1 (type 1); F- at inferior and F+ at V, (type 2); and f-/F+ at inferior and P+ at V1 (type 3). Reverse typical AFL showed 2 types flutter wave morphologies comprised of F+ at inferior and F- at V, (type 1); and P+ at inferior and isoelectric at V1 (type 2). However, there were no significant different of right atrial wavefront activations between those AFL morphologies types. Ninety percent of CTI dependent AFL demonstrated no transversal conduction at crista terminalis, on the contrary all ULR demonstrated transversal conduction. During ULR, CVL was faster than CVT (1.23 ± 0.43 vs. 0.73 ± 0.30 m/sec, p < 0.00l). The ratio of CVL/CVt (1.95 :t 0.77) had inverse correlation with the gap width (1.57 ± 6.8 mm) (p < 0.001). A new diagnostic algorithm based on morphology and amplitude of flutter wave at lead I had accuracy of 90 to 97%, sensitivity of 82 to 100% and specificity of 95% to differentiate reverse typical AFL from ULR. The majority of focal AT originated hom crista terminalis and showed a preferential wavefront conduction before spreading to the whole atrium. The success rate of radiofrequency ablation of CTI dependent AFL, crista terminalis gap of ULR and focal AT were 96%, 90% and 91.7% respectively.

Conclusion: Typical AFL is the predominant AFL cases and majority of AFL had structural heart disease. There was no right atrial activation different among flutter wave morphology types of CTI dependent AFL. The majority of crista tenninalis gap was functional and always exists during ULR. A new diagnostic ECG algorithm has been demonstrated to have excellent accuracy to differentiate typical AFL from ULR. The wavefront of focal AT spreads out to the whole atrium after traveled in preferential conduction. RPA was effective to eliminate CTI and non-CTI dependent AFL, and focal AT.
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